

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 14:47:27 ON 12 SEP 2005

=> file ca

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'CA' ENTERED AT 14:47:33 ON 12 SEP 2005

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FILE LAST UPDATED: 8 Sep 2005 (20050908/ED)

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=> e 102:179661/an

E1	1	102:17966/AN
E2	1	102:179660/AN
E3	1 -->	102:179661/AN
E4	1	102:179662/AN
E5	1	102:179663/AN
E6	1	102:179664/AN
E7	1	102:179665/AN
E8	1	102:179666/AN
E9	1	102:179667/AN
E10	1	102:179668/AN
E11	1	102:179669/AN
E12	1	102:17967/AN

=> s e3

L1 1 "102:179661"/AN

=> d

L1 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AN 102:179661 CA

TI Norepinephrine amplifies human chorionic gonadotropin-stimulated androgen biosynthesis by ovarian theca-interstitial cells

AU Dyer, Cheryl A.; Erickson, Gregory F.

CS Dep. Reprod. Med., Univ. California, San Diego, La Jolla, CA, 92093, USA

SO Endocrinology (1985), 116(4), 1645-52

CODEN: ENDOAO; ISSN: 0013-7227

DT Journal

LA English

=> e 97:157029/an

E1	1	97:157027/AN
E2	1	97:157028/AN
E3	1 -->	97:157029/AN
E4	1	97:15703/AN
E5	1	97:157030/AN
E6	1	97:157031/AN
E7	1	97:157032/AN
E8	1	97:157033/AN
E9	1	97:157034/AN
E10	1	97:157035/AN
E11	1	97:157036/AN
E12	1	97:157037/AN

=> s e3

L2 1 "97:157029"/AN

=> d

L2 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AN 97:157029 CA

TI Catecholamine stimulation of androgen production by mouse interstitial cells in primary culture

AU Moger, William H.; Murphy, Paul R.; Casper, Robert F.

CS Dep. Physiol. Biophys., Dalhousie Univ., Halifax, NS, Can.

SO Journal of Andrology (1982), 3(4), 227-31

CODEN: JOAND3; ISSN: 0196-3635

DT Journal

LA English

=> d ab 11

L1 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AB Ovarian theca-interstitial cells, when cultured in serum-free medium, secreted androgens in response to human chorionic gonadotropin (hCG) [9002-61-3] stimulation. This production was dependent on time (maximum production

attained after 96 h) and dose (half-maximal ED of hCG, 9 ng/mL). When the sympathomimetics norepinephrine [51-41-2], epinephrine [51-43-4], and isoproterenol [7683-59-2] were added to the medium, androgen production in response to hCG was enhanced by 100-300%. The ability of the catecholamines to stimulate androgen production was dependent on the continuous presence of hCG. Treatment with catecholamines alone did not induce theca-interstitial cells to produce androgens. Catecholamine stimulation of steroid hormone metabolism was selective for intermediates in the  $\Delta 4$ -pathway, with greatest increases occurring in the production of androstenedione [63-05-8] and testosterone [58-22-0]. The effect of the catecholamines on androgen production was dependent on both  $\beta 1$ - and  $\beta 2$ -adrenergic receptors. The acquisition of catecholamine responsiveness was specific to hCG; if theca-interstitial cells were induced to differentiate with either PGE2 or cholera toxin, then isoproterenol did not enhance androgen synthesis. The catecholamine-induced increases in androgen production were not due to a granulosa cell contribution of steroid. The interstitial cells are the only steroid-producing cells in the ovary that are directly innervated by norepinephrine-containing fibers of the sympathetic nervous system. This catecholamine-augmented androgen production provides a direct link between the autonomic nervous system and regulation of ovarian steroid synthesis.

=> d ab 12

L2 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AB The catecholamines 1-isoproterenol [2964-04-7], 1-epinephrine [51-43-4], and (-)-norepinephrine [51-41-2] stimulated androgen production by mouse interstitial cells in primary culture. The amount of androgen produced in response to maximum stimulation with these amines was less than that produced with maximum human chorionic gonadotropin (hCG) [9002-61-3] stimulation, but produced an additive effect when combined with a submaximal concentration of

hCG.

The stimulatory effect of isoproterenol was blocked by the  $\beta$ -receptor antagonist propranolol. Isoproterenol did not stimulate androgen production by either freshly isolated mouse interstitial cells or whole decapsulated testes.

=> e 97:36514/an

E1	1	97:36512/AN
E2	1	97:36513/AN
E3	1 -->	97:36514/AN
E4	1	97:36515/AN
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E8	1	97:36519/AN
E9	1	97:3652/AN
E10	1	97:36520/AN
E11	1	97:36521/AN
E12	1	97:36522/AN

=> s e3

L3 1 "97:36514"/AN

=> d

L3 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AN 97:36514 CA

TI Effect of adrenotropic substances on the growth and maturation of oocytes of the sea urchin, *Strongylocentrotus nudus*

AU Khotimchenko, Yu. S.

CS Far East Sci. Cent., Inst. Mar. Biol., Vladivostok, 690022, USSR

SO Experientia (1982), 38(6), 696-7

CODEN: EXPEAM; ISSN: 0014-4754

DT Journal

LA English

=> d ab

L3 ANSWER 1 OF 1 CA COPYRIGHT 2005 ACS on STN

AB The oocyte size was decreased after the administration of the adrenomimetics, noradrenaline, dopamine, and ephedrine, to *S. nudus*. The administration of the adrenolytics, propranolol, oxyprenolol, and dihydroergotamine, caused an increase in the sea urchin oocyte size. Thus, oogenesis in the sea urchin may be regulated by a monoaminergic system.

=> log y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
15.51	15.72

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-2.04	-2.04

CA SUBSCRIBER PRICE

STN INTERNATIONAL LOGOFF AT 14:50:53 ON 12 SEP 2005

aerobic conditions. Within a 2 h time interval, leukocyte phagocytosis in the presence of these vasoactive compounds resulted in a reduction in bacterial count in the supernatant. Intracellular kill of the phagocytized bacteria, as exemplified by the reduction in the total count of the suspensions, was comparable in the suspensions containing vasoactive compounds and the controls.

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria, Eubacteria, Microorganisms

ORGN Classifier

Leporidae 86040

Super Taxa

Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

AB In rabbits, epinephrine altered the distribution of the i.v. injected fluorescein dye. At the epinephrine injection site the skin did not fluoresce, indicating that the dye had limited access to that tissue. The uninjected skin as well as the skin subjected to phentolamine fluoresced brightly. The minimal concentration of a phentolamine solution that inhibited epinephrine (1:100,000)-induced vasoconstriction was 1:50,000. Epinephrine potentiated the development of infection. When 6.0 + 106 Staphylococcus aureus were injected into skin in the presence of epinephrine, all sites developed infection as compared to only a 12.5% infection rate in the control wounds. The addition of phentolamine (1:50,000) to the epinephrine eliminated its damaging effects. The infection rate of contaminated tissue subjected to the mixture of vasoactive drugs did not differ significantly from that of controls. Phentolamine by itself did not enhance the incidence of infection. The infection rate of tissues was proportional to the magnitude of wound induration and the number of viable bacteria recovered from the wounds. Under aerobic conditions, the presence of epinephrine and phentolamine in the nutrient broth did not influence the growth of S. aureus in vitro. The bacterial counts of the suspensions containing the vasoactive compounds did not differ significantly from those of the control suspensions. Epinephrine and phentolamine did not interfere with in vitro leukocyte phagocytosis of bacteria and subsequent intracellular kill under aerobic conditions. Within a 2 h time interval, leukocyte phagocytosis in the presence of these vasoactive compounds resulted in a reduction in bacterial count in the supernatant. Intracellular kill of the phagocytized bacteria, as exemplified by the reduction in the total count of the suspensions, was comparable in the suspensions containing vasoactive compounds and the controls.

L13 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN

TI Effect of copper on thyroxine potentiation of in vitro epinephrine action on smooth muscle

AN 1964:11057 CAPLUS

DN 60:11057

OREF 60:2016e-g

TI Effect of copper on thyroxine potentiation of in vitro epinephrine action on smooth muscle

AU Shida, H.; Meyers, M. A.; Barker, S. B.

CS Univ. of Alabama Med. Center, Birmingham

SO Journal of Pharmacology and Experimental Therapeutics (1963), 141(3), 280-4

CODEN: JPETAB; ISSN: 0022-3565

DT Journal

LA Unavailable

TI Effect of copper on thyroxine potentiation of in vitro epinephrine action on smooth muscle  
 SO Journal of Pharmacology and Experimental Therapeutics (1963), 141(3), 280-4  
 CODEN: JPETAB; ISSN: 0022-3565  
 AB Using the method of Furchgott and Bhadrakom (CA 47, 10111i) employing the contraction of a helical aortic strip of a rabbit, it was confirmed that in vitro addition of thyroxine (I) enhanced the contracting effect of epinephrine and norepinephrine. The KrebsRinger NaHCO3 glucose bath solution contained varying contaminating levels of Cu. No enhancing effect was observed at low Cu levels (1.5  $\gamma$ /l.) but was clearly evident at higher levels (3.5 and 6.6  $\gamma$ /l.). Since ethylenediaminetetraacetate behaved similarly to I, the effect was probably due to chelation of the Cu in the working solution Zn and Fe salts in higher concentration (100  $\gamma$ /l.) also suppressed catechol amine action which could be reversed by I. Mn at 10  $\gamma$ /l. was as effective as Cu but was not reversed by I.

IT Viruses  
 (poliomyelitis, ribonucleic acid of, base composition of)  
 AB Using the method of Furchgott and Bhadrakom (CA 47, 10111i) employing the contraction of a helical aortic strip of a rabbit, it was confirmed that in vitro addition of thyroxine (I) enhanced the contracting effect of epinephrine and norepinephrine. The KrebsRinger NaHCO3 glucose bath solution contained varying contaminating levels of Cu. No enhancing effect was observed at low Cu levels (1.5  $\gamma$ /l.) but was clearly evident at higher levels (3.5 and 6.6  $\gamma$ /l.). Since ethylenediaminetetraacetate behaved similarly to I, the effect was probably due to chelation of the Cu in the working solution Zn and Fe salts in higher concentration (100  $\gamma$ /l.) also suppressed catechol amine action which could be reversed by I. Mn at 10  $\gamma$ /l. was as effective as Cu but was not reversed by I.

L13 ANSWER 7 OF 10 MEDLINE on STN  
 TI Herpes simplex virus recovery in neural tissues after ocular HSV shedding induced by epinephrine iontophoresis to the rabbit cornea.  
 AN 83134751 MEDLINE  
 DN PubMed ID: 6298139  
 TI Herpes simplex virus recovery in neural tissues after ocular HSV shedding induced by epinephrine iontophoresis to the rabbit cornea.  
 AU Hill J M; Kwon B S; Shimomura Y; Colborn G L; Yaghmai F; Gangarosa L P  
 NC NEI-EY-03331 (NEI)  
 NIDR-DE-04917 (NIDCR)  
 SO Investigative ophthalmology & visual science, (1983 Feb) 24 (2) 243-7.  
 Journal code: 7703701. ISSN: 0146-0404.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198304  
 ED Entered STN: 19900318  
 Last Updated on STN: 20000303  
 Entered Medline: 19830415  
 TI Herpes simplex virus recovery in neural tissues after ocular HSV shedding induced by epinephrine iontophoresis to the rabbit cornea.  
 SO Investigative ophthalmology & visual science, (1983 Feb) 24 (2) 243-7.  
 Journal code: 7703701. ISSN: 0146-0404.  
 AB Ocular HSV-1 shedding from latently infected rabbits was induced by iontophoresis of 0.01% epinephrine into the eye. Anodal Iontophoresis of epinephrine was performed at 0.8 mAmps for 8 min once a day for 3

consecutive days. Shedding was determined by the presence of HSV-1 in the preocular tear film obtained via eye swabs. Bilateral epinephrine iontophoresis performed on selected days during 220-280 days after inoculation resulted in HSV-1 shedding in 75% of the eyes (30/40) and 100% of the rabbits (20/20). Following the induction of ocular HSV-1 shedding, rabbits were killed and selected neural tissues were homogenized. Cell-free preparations were assayed for the presence of infectious virions using primary rabbit kidney cell monolayers. When the tissues were homogenized immediately after death, virus was detected in only one neural tissue, the trigeminal ganglia. However, when the tissues were incubated in vitro for 18-24 hours prior to the homogenization, infectious HSV-1 was recovered from homogenates of the trigeminal ganglion, superior cervical ganglion, the ophthalmic branch of the trigeminal nerve, and the root entry zone of the trigeminal nerve. A relationship was noted between the time of the last ocular shedding and recovery of infectious HSV from the tissue homogenates. Furthermore, a positive correlation in 11 eyes between the recovery of HSV-1 from the preocular tear film and HSV-1 recovery from one or more corresponding neural tissues was found. These results suggested that epinephrine iontophoresis to the cornea triggered an "alteration" in the state of the virus in the neural tissues of the latently infected rabbits and that the change can be related to the induced ocular shedding.

CT Animals

Cornea: MI, microbiology

Culture Techniques

\*Epinephrine: AD, administration & dosage

\*Iontophoresis

\*Keratitis, Dendritic: MI, microbiology

\*Nerve Tissue: MI, microbiology

Rabbits

Research Support, U.S. Gov't, P.H.S.

\*Simplexvirus: GD, growth & development

Tears: MI, microbiology

Time Factors

Virion: IP, isolation & purification

\*Virus Activation

AB Ocular HSV-1 shedding from latently infected rabbits was induced by iontophoresis of 0.01% epinephrine into the eye. Anodal Iontophoresis of epinephrine was performed at 0.8 mAmps for 8 min once a day for 3 consecutive days. Shedding was determined by the presence of HSV-1 in the preocular tear film obtained via eye swabs. Bilateral epinephrine iontophoresis performed on selected days during 220-280 days after inoculation resulted in HSV-1 shedding in 75% of the eyes (30/40) and 100% of the rabbits (20/20). Following the induction of ocular HSV-1 shedding, rabbits were killed and selected neural tissues were homogenized. Cell-free preparations were assayed for the presence of infectious virions using primary rabbit kidney cell monolayers. When the tissues were homogenized immediately after death, virus was detected in only one neural tissue, the trigeminal ganglia. However, when the tissues were incubated in vitro for 18-24 hours prior to the homogenization, infectious HSV-1 was recovered from homogenates of the trigeminal ganglion, superior cervical ganglion, the ophthalmic branch of the trigeminal nerve, and the root entry zone of the trigeminal nerve. A relationship was noted between the time of the last ocular shedding and recovery of infectious HSV from the tissue homogenates. Furthermore, a positive correlation in 11 eyes between the recovery of HSV-1 from the preocular tear film and HSV-1 recovery from one or more corresponding neural tissues was found. These results suggested that epinephrine iontophoresis to the cornea triggered an "alteration" in the state of the virus in the neural tissues of the latently infected rabbits and that the change can be related to the induced ocular shedding.

L13 ANSWER 8 OF 10 USPATFULL on STN

TI Method employing gonadal hormones and dopamine agonist  
intended for combined use in the improvement of lymphocyte function  
AN 90:98709 USPATFULL  
TI Method employing gonadal hormones and dopamine agonist  
intended for combined use in the improvement of lymphocyte function  
IN Smith, R. Arnold, Jackson, MS, United States  
PA McAdory, George D., Jackson, MS, United States (U.S. individual)  
PI US 4980358 19901225 <--  
AI US 1990-494327 19900316 (7)  
RLI Continuation-in-part of Ser. No. US 1988-177121, filed on 4 Apr 1988,  
now patented, Pat. No. US 4929640  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Friedman, Stanley J.; Assistant Examiner: Criares, T.  
J.  
LREP Epstein, Edell & Retzer  
CLMN Number of Claims: 31  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 498  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 9 OF 10 USPATFULL on STN

TI Dopamine- $\beta$ -hydroxylase inhibitors  
AN 88:48763 USPATFULL  
TI Dopamine- $\beta$ -hydroxylase inhibitors  
IN Finkelstein, Joseph A., Philadelphia, PA, United States  
Kruse, Lawrence I., Haddonfield, NJ, United States  
Leonard, Thomas B., Haverford, PA, United States  
PA SmithKline Beckman Corporation, Philadelphia, PA, United States (U.S.  
corporation)  
PI US 4761415 19880802 <--  
AI US 1986-901120 19860828 (6)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Gerstl, Robert; Assistant Examiner: Shen, Cecilia  
LREP Fabiano, Vincent L., Suter, Stuart R., Lourie, Alan D.  
CLMN Number of Claims: 15  
ECL Exemplary Claim: 1,11,15  
DRWN No Drawings  
LN.CNT 562  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 10 OF 10 USPATFULL on STN

TI Dopamine- $\beta$ -hydroxylase inhibitors  
AN 86:69756 USPATFULL  
TI Dopamine- $\beta$ -hydroxylase inhibitors  
IN Finkelstein, Joseph A., Philadelphia, PA, United States  
Kaiser, Carl, Haddon Heights, NJ, United States  
Kruse, Lawrence I., Haddonfield, NJ, United States  
PA SmithKlein Beckman Corporation, Philadelphia, PA, United States (U.S.  
corporation)  
PI US 4628059 19861209 <--  
AI US 1985-793513 19851031 (6)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Bond, Robert T.  
LREP Fabiano, Vincent L., Suter, Stuart R., Lourie, Alan D.  
CLMN Number of Claims: 21  
ECL Exemplary Claim: 1,15  
DRWN No Drawings  
LN.CNT 604



CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> log hold

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

69.16

203.62

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

CA SUBSCRIBER PRICE

-0.73

-2.77

SESSION WILL BE HELD FOR 60 MINUTES

STN INTERNATIONAL SESSION SUSPENDED AT 13:55:48 ON 12 SEP 2005

were preincubated with melanin, thus suggesting that interaction of melanin with viral proteins is an important aspect of the antiviral mechanism. These results make synthetic soluble melanins interesting candidates for further study as possible anti-HIV-1 therapeutics.

L13 ANSWER 3 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 3  
TI MANGANESE II UNCOUPLING OF THE CATECHOLAMINE SENSITIVE ADENYLATE  
CYCLASE SYSTEM OF RAT RETICULOCYTES PARALLEL EFFECTS ON CHOLERA TOXIN  
CATALYZED ADP RIBOSYLATION OF THE SYSTEM  
AN 1982:68444 TOXCENTER  
CP Copyright (c) 2005 The Thomson Corporation  
DN PREV198273070979  
TI MANGANESE II UNCOUPLING OF THE CATECHOLAMINE SENSITIVE ADENYLATE  
CYCLASE SYSTEM OF RAT RETICULOCYTES PARALLEL EFFECTS ON CHOLERA TOXIN  
CATALYZED ADP RIBOSYLATION OF THE SYSTEM  
AU LIMBIRD L E [Reprint author]; MACMILLAN S T  
CS DEP PHARMACOL, SCH MED, VANDERBILT UNIV, NASHVILLE, TENN 37232, USA  
SO Biochimica et Biophysica Acta, (1981) Vol. 677, No. 3-4, pp.  
408-416.  
CODEN: BBACAQ. ISSN: 0006-3002.  
DT Article  
FS BIOSIS  
OS BIOSIS 1982:210995  
LA ENGLISH  
ED Entered STN: 20011116  
Last Updated on STN: 20011116  
TI MANGANESE II UNCOUPLING OF THE CATECHOLAMINE SENSITIVE ADENYLATE  
CYCLASE SYSTEM OF RAT RETICULOCYTES PARALLEL EFFECTS ON CHOLERA TOXIN  
CATALYZED ADP RIBOSYLATION OF THE SYSTEM  
SO Biochimica et Biophysica Acta, (1981) Vol. 677, No. 3-4, pp.  
408-416.  
CODEN: BBACAQ. ISSN: 0006-3002.  
AB High concentrations of Mn<sup>2+</sup> interfere with functional interactions between  
the GTP-binding regulatory protein (G) and the catalytic moiety (C) of  
adenylate cyclase without perturbing interactions between receptor (R) and  
component G in rat reticulocyte membranes. The ability of cholera toxin  
to ADP-ribosylate component G and to enhance GTP-stimulated  
adenylate cyclase activity also appears to be correlated with the efficacy  
of the communication of component G with the adenylate cyclase system.  
Thus, increasing the concentration of Mn<sup>2+</sup> in rat reticulocyte membrane  
during in vitro incubations causes a parallel loss of  
Gpp(NH)p-stimulated adenylate cyclase activity, cholera toxin-catalyzed  
[32P]ADP-ribosylation of the 42,000 MW subunit of component G and cholera  
toxin-catalyzed enhancement of GTP-sensitive adenylate cyclase activity.  
Removal of Mn<sup>2+</sup> by washing the membranes completely restores the  
sensitivity of adenylate cyclase to all effectors, including cholera  
toxin. Exposure of membranes to Mn<sup>2+</sup> apparently provides a useful tool  
for reversibly uncoupling catecholamine-sensitive adenylate cyclase  
systems. The extent of cholera toxin-catalyzed ADP-ribosylation of  
membrane substrates, i.e., the G component may rely on functional  
communication among the various components of the adenylate cyclase  
system. A corollary of the latter is that the amount of  
[32P]ADP-ribose-product detected in a membrane may reflect both the  
quantity and coupling efficiency of component G.  
ORGN Classifier  
Vibrionaceae 06704  
Super Taxa  
Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;  
Microorganisms  
Taxa Notes  
Bacteria, Eubacteria, Microorganisms  
ORGN Classifier  
Muridae 86375  
Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,  
Rodents, Vertebrates

- AB High concentrations of  $Mn^{2+}$  interfere with functional interactions between the GTP-binding regulatory protein (G) and the catalytic moiety (C) of adenylate cyclase without perturbing interactions between receptor (R) and component G in rat reticulocyte membranes. The ability of cholera toxin to ADP-ribosylate component G and to enhance GTP-stimulated adenylate cyclase activity also appears to be correlated with the efficacy of the communication of component G with the adenylate cyclase system. Thus, increasing the concentration of  $Mn^{2+}$  in rat reticulocyte membrane during *in vitro* incubations causes a parallel loss of Gpp(NH)p-stimulated adenylate cyclase activity, cholera toxin-catalyzed [ $^{32}P$ ]ADP-ribosylation of the 42,000 MW subunit of component G and cholera toxin-catalyzed enhancement of GTP-sensitive adenylate cyclase activity. Removal of  $Mn^{2+}$  by washing the membranes completely restores the sensitivity of adenylate cyclase to all effectors, including cholera toxin. Exposure of membranes to  $Mn^{2+}$  apparently provides a useful tool for reversibly uncoupling catecholamine-sensitive adenylate cyclase systems. The extent of cholera toxin-catalyzed ADP-ribosylation of membrane substrates, i.e., the G component may rely on functional communication among the various components of the adenylate cyclase system. A corollary of the latter is that the amount of [ $^{32}P$ ]ADP-ribose-product detected in a membrane may reflect both the quantity and coupling efficiency of component G.

L13 ANSWER 4 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 4

TI EFFECTS OF DOPAMINE ON PROLACTIN SECRETION AND CYCLIC AMP  
ACCUMULATION IN THE RAT ANTERIOR PITUITARY GLAND

AN 1981:69281 TOXCENTER

CP Copyright (c) 2005 The Thomson Corporation

DN PREV198171082911

TI EFFECTS OF DOPAMINE ON PROLACTIN SECRETION AND CYCLIC AMP  
ACCUMULATION IN THE RAT ANTERIOR PITUITARY GLAND

AU RAY K P [Reprint author]; WALLIS M

CS SCH BIOL SCI, UNIV SUSSEX, FALMER, BRIGHTON BN1 9QG, ENGL, UK

SO Biochemical Journal, (1981) Vol. 194, No. 1, pp. 119-128.

ISSN: 0264-6021.

DT Article

FS BIOSIS

OS BIOSIS 1981:212919

LA ENGLISH

ED Entered STN: 20011116

Last Updated on STN: 20011116

TI EFFECTS OF DOPAMINE ON PROLACTIN SECRETION AND CYCLIC AMP  
ACCUMULATION IN THE RAT ANTERIOR PITUITARY GLAND

SO Biochemical Journal, (1981) Vol. 194, No. 1, pp. 119-128.

ISSN: 0264-6021.

- AB The effects of dopamine on pituitary prolactin secretion and pituitary cAMP accumulation were studied by using anterior pituitary glands from adult female rats, incubated *in vitro*. During 2 h incubations, significant inhibition of prolactin secretion was achieved at concentrations between 1 and 10 nM-dopamine. However, 0.1-1  $\mu M$ -dopamine was required before a significant decrease in pituitary cAMP content was observed. In the presence of 1  $\mu M$ -dopamine, pituitary cAMP content decreased rapidly to reach about 75% of the control value within 20 min, and there was no further decrease for at least 2 h. Incubation with the phosphodiesterase inhibitors, theophylline (8 mM) or isobutylmethylxanthine (2mM), increased pituitary cAMP concentrations 3- and 6-fold, respectively. Dopamine (1  $\mu M$ ) had no effect on the cAMP accumulation measured in the presence of theophylline, but inhibited the isobutylmethylxanthine-induced increase by 50%. The dopamine inhibition of prolactin secretion was not affected by either inhibitor. Two

- TI Novel nerve growth factor-responsiveness of catecholamine biosynthesis and secretion in clonal rat pheochromocytoma cells cultured in a hormone-supplemented serum-free medium
- L9 ANSWER 34 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Plasticity of pheochromocytoma (PC12) cells demonstrated by nerve growth factor or glucocorticoid treatment: a catecholamine fluorescence and electron microscopic investigation
- L9 ANSWER 35 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Immunohistochemical and immunocytochemical localization of myosin, chromogranin A and dopamine- $\beta$ -hydroxylase in nerve cells in culture and in adrenal glands
- L9 ANSWER 36 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Biochemical characterization of norepinephrine-3H uptake in dissociated brain cell cultures from chick embryos
- L9 ANSWER 37 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI A clonal rat pheochromocytoma cell line possesses synthesizing ability of  $\gamma$ -aminobutyric acid together with catecholamine and acetylcholine
- L9 ANSWER 38 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Catecholamine-containing neurons from rat brain in culture: response to peripheral and central target tissues
- L9 ANSWER 39 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Role of nerve growth factor in the development of rat sympathetic neurons in vitro. I. Survival, growth, and differentiation of catecholamine production
- L9 ANSWER 40 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Selective induction of tyrosine hydroxylase and dopamine  $\beta$ -hydroxylase by nerve growth factor: comparison between adrenal medulla and sympathetic ganglia of adult and newborn rats
- L9 ANSWER 41 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Changes in catecholamine synthesizing enzyme activities during neuronal growth and degeneration
- L9 ANSWER 42 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Production of catecholamine-containing cells in vitro by young chick embryos studied by the histochemical fluorescence method
- L9 ANSWER 43 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Production of catecholamine-containing cells in vitro by young chick embryos. Effects of nerve growth factor (NGF) and its antiserum
- L9 ANSWER 44 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Influence of pituitary hormones and norepinephrine on the size of adipose cells in organ culture

=> d ti bib hit ab 23,24, 28

- L9 ANSWER 23 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Catecholamine modulation of embryonic palate mesenchymal cell DNA synthesis  
AN 104:82581 CA  
TI Catecholamine modulation of embryonic palate mesenchymal cell DNA synthesis

AU Pisano, M. Michele; Schneiderman, Martin H.; Greene, Robert M.  
 CS Daniel Baugh Inst., Thomas Jefferson Univ., Philadelphia, PA, 19107, USA  
 SO Journal of Cellular Physiology (1986), 126(1), 84-92  
 CODEN: JCLLAX; ISSN: 0021-9541

DT Journal  
 LA English

TI Catecholamine modulation of embryonic palate mesenchymal cell DNA synthesis

SO Journal of Cellular Physiology (1986), 126(1), 84-92  
 CODEN: JCLLAX; ISSN: 0021-9541

AB By utilizing quiescent populations of murine embryonic palate mesenchymal (MEPM) cells in vitro, hormonal regulation of palatal cell proliferation was examined. MEPM cells in culture were rendered quiescent by 48 h serum deprivation and were subsequently released from growth arrest by readdn. of medium containing 10% (volume/volume) serum. The progression of cells into S-phase of the cell cycle was monitored by autoradiog. anal. of tritiated thymidine incorporation. Palate mesenchymal cell entry into S-phase was preceded by a 6-8-h prereplicative lag period, after which time DNA synthesis increased and cells reached a maximum labeling index by 22 h. Addition of 10  $\mu$ M ( $\pm$ )-isoproterenol [149-53-1] to cell cultures at the time of release from growth arrest lengthened the prereplicative lag period and delayed cellular entry into S-phase by an addnl. 2-4 h. The rate of cellular progression through S-phase remained unaltered. The inhibitory effect of isoproterenol on the initiation of MEPM cell DNA synthesis was abolished by pretreatment of cells with propranolol at a concentration (100  $\mu$ M) that prevented isoproterenol-induced elevations of cAMP [60-92-4]. Addition of PGE2 [363-24-6] to cell cultures, at a concentration that markedly stimulates cAMP formation, mimicked the inhibitory effect of isoproterenol on cellular progression into S-phase. Evidently, the  $\beta$ -adrenergic catecholamine isoproterenol modulates MEPM cell proliferation in vitro via a receptor-mediated mechanism, and the delayed initiation of DNA synthesis in these cells is a cAMP-dependent phenomenon.

IT Cell cycle  
 (S-phase, DNA formation by palate of embryo response to catecholamine in relation to)

AB By utilizing quiescent populations of murine embryonic palate mesenchymal (MEPM) cells in vitro, hormonal regulation of palatal cell proliferation was examined. MEPM cells in culture were rendered quiescent by 48 h serum deprivation and were subsequently released from growth arrest by readdn. of medium containing 10% (volume/volume) serum. The progression of cells into S-phase of the cell cycle was monitored by autoradiog. anal. of tritiated thymidine incorporation. Palate mesenchymal cell entry into S-phase was preceded by a 6-8-h prereplicative lag period, after which time DNA synthesis increased and cells reached a maximum labeling index by 22 h. Addition of 10  $\mu$ M ( $\pm$ )-isoproterenol [149-53-1] to cell cultures at the time of release from growth arrest lengthened the prereplicative lag period and delayed cellular entry into S-phase by an addnl. 2-4 h. The rate of cellular progression through S-phase remained unaltered. The inhibitory effect of isoproterenol on the initiation of MEPM cell DNA synthesis was abolished by pretreatment of cells with propranolol at a concentration (100  $\mu$ M) that prevented isoproterenol-induced elevations of cAMP [60-92-4]. Addition of PGE2 [363-24-6] to cell cultures, at a concentration that markedly stimulates cAMP formation, mimicked the inhibitory effect of isoproterenol on cellular progression into S-phase. Evidently, the  $\beta$ -adrenergic catecholamine isoproterenol modulates MEPM cell proliferation in vitro via a receptor-mediated mechanism, and the delayed initiation of DNA synthesis in these cells is a cAMP-dependent phenomenon.

L9 ANSWER 24 OF 44 CA COPYRIGHT 2005 ACS on STN  
 TI Relationship between dopamine content and its secretion in PC12

cells as a function of cell growth

AN 103:207450 CA

TI Relationship between dopamine content and its secretion in PC12 cells as a function of cell growth

AU Takashima, Akihiko; Koike, Tatsuro

CS Dep. Nat. Sci., Saga Med. Sch., Nabeshima, 840-01, Japan

SO Biochimica et Biophysica Acta (1985), 847(1), 101-7  
CODEN: BBACAQ; ISSN: 0006-3002

DT Journal

LA English

TI Relationship between dopamine content and its secretion in PC12 cells as a function of cell growth

SO Biochimica et Biophysica Acta (1985), 847(1), 101-7  
CODEN: BBACAQ; ISSN: 0006-3002

AB The relation between dopamine [51-61-6] biosynthesis and its stimulus-induced secretion was studied in PC12 cells as a function of cell growth. The endogenous dopamine content depended on cell growth, and reached a maximum in the stationary phase. This increase was associated both with an increase in the specific activity of tyrosine 3-monooxygenase [9036-22-0], and with an increase of DOPA-decarboxylase [9042-64-2] in the cells. On the other hand, the maximal release of dopamine occurred in the late exponential phase before the endogenous dopamine was maximally synthesized in the cells. Moreover, the uptake of  $45\text{Ca}^{2+}$  stimulated with either carbamylcholine [462-58-8] or high  $\text{K}^+$  was also regulated by cell division: the maximal uptake took place in the same period of culture in which the maximal release of dopamine was observed. Thus, the biosynthesis and secretion of dopamine are sep. regulated in PC12 cells.

ST pheochromocytoma dopamine metab cell division

IT Biological transport  
(of calcium, by pheochromocytoma, cell division and dopamine formation and release in relation to)

IT Pheochromocytoma  
(PC12, dopamine formation and secretion by, cell division in relation to)

IT Cell division  
(mitosis, by pheochromocytoma, dopamine formation and release in relation to)

IT 462-58-8 7440-09-7, biological studies  
RL: BIOL (Biological study)  
(calcium uptake by pheochromocytoma stimulation by, cell division and dopamine release in relation to)

IT 51-61-6, biological studies  
RL: BIOL (Biological study)  
(formation and secretion of, by pheochromocytoma, cell division in relation to)

IT 7440-70-2, biological studies  
RL: BIOL (Biological study)  
(uptake of, by pheochromocytoma, cell division and dopamine release in relation to)

AB The relation between dopamine [51-61-6] biosynthesis and its stimulus-induced secretion was studied in PC12 cells as a function of cell growth. The endogenous dopamine content depended on cell growth, and reached a maximum in the stationary phase. This increase was associated both with an increase in the specific activity of tyrosine 3-monooxygenase [9036-22-0], and with an increase of DOPA-decarboxylase [9042-64-2] in the cells. On the other hand, the maximal release of dopamine occurred in the late exponential phase before the endogenous dopamine was maximally synthesized in the cells. Moreover, the uptake of  $45\text{Ca}^{2+}$  stimulated with either carbamylcholine [462-58-8] or high  $\text{K}^+$  was also regulated by cell division: the maximal uptake took place in the same period of culture in which the maximal release of dopamine was observed. Thus, the biosynthesis and secretion of dopamine are sep. regulated in PC12 cells.

of prolactin secretion was not affected by either inhibitor. Two derivatives of cAMP (dibutyryl cAMP and 8-bromo cAMP) were unable to block the dopamine (1  $\mu$ M) inhibition of prolactin secretion, although 8-bromo cAMP (2mM) significantly stimulated prolactin secretion and both compounds increased growth hormone [GH] release. Cholera toxin (3  $\mu$ g/ml for 4 h) increased pituitary cAMP concentrations 4-to 5-fold, but had no effect on prolactin secretion. The inhibition of prolactin secretion by dopamine was unaffected by cholera toxin, despite the fact that dopamine had no effect on the raised pituitary cAMP concentration caused by this factor. Dopamine had no significant effect on either basal or stimulated GH secretion under any of the conditions tested. The inhibitory effects of dopamine on prolactin secretion are probably not mediated by lowering of cAMP concentration, although modulation of the concentration of this nucleotide, in some other circumstances, may alter the secretion of the hormone.

L13 ANSWER 5 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 5  
 TI STUDIES OF THE MECHANISMS BY WHICH EPINEPHRINE DAMAGES TISSUE DEFENSES  
 AN 1978:55907 TOXCENTER  
 CP Copyright (c) 2005 The Thomson Corporation  
 DN PREV197865074388  
 TI STUDIES OF THE MECHANISMS BY WHICH EPINEPHRINE DAMAGES TISSUE DEFENSES  
 AU MAGEE C [Reprint author]; RODEHEAVER G T; EDGERTON M T; GOLDEN G T; HAURY B; EDLICH R F  
 CS DEP PLAST SURG, UNIV VA SCH MED, CHARLOTTESVILLE, VA 22901, USA  
 SO Journal of Surgical Research, (1977) Vol. 23, No. 2, pp. 126-131.  
 CODEN: JSGRA2. ISSN: 0022-4804.  
 DT Article  
 FS BIOSIS  
 OS BIOSIS 1978:187388  
 LA ENGLISH  
 ED Entered STN: 20011116  
 Last Updated on STN: 20011116  
 TI STUDIES OF THE MECHANISMS BY WHICH EPINEPHRINE DAMAGES TISSUE DEFENSES  
 SO Journal of Surgical Research, (1977) Vol. 23, No. 2, pp. 126-131.  
 CODEN: JSGRA2. ISSN: 0022-4804.  
 AB In rabbits, epinephrine altered the distribution of the i.v. injected fluorescein dye. At the epinephrine injection site the skin did not fluoresce, indicating that the dye had limited access to that tissue. The uninjected skin as well as the skin subjected to phentolamine fluoresced brightly. The minimal concentration of a phentolamine solution that inhibited epinephrine (1:100,000)-induced vasoconstriction was 1:50,000. Epinephrine potentiated the development of infection. When  $6.0 \times 10^6$  Staphylococcus aureus were injected into skin in the presence of epinephrine, all sites developed infection as compared to only a 12.5% infection rate in the control wounds. The addition of phentolamine (1:50,000) to the epinephrine eliminated its damaging effects. The infection rate of contaminated tissue subjected to the mixture of vasoactive drugs did not differ significantly from that of controls. Phentolamine by itself did not enhance the incidence of infection. The infection rate of tissues was proportional to the magnitude of wound induration and the number of viable bacteria recovered from the wounds. Under aerobic conditions, the presence of epinephrine and phentolamine in the nutrient broth did not influence the growth of S. aureus in vitro. The bacterial counts of the suspensions containing the vasoactive compounds did not differ significantly from those of the control suspensions. Epinephrine and phentolamine did not interfere with in vitro leukocyte phagocytosis of bacteria and subsequent intracellular kill under

derivatives of cAMP (dibutyryl cAMP and 8-bromo cAMP) were unable to block the dopamine (1  $\mu$ M) inhibition of prolactin secretion, although 8-bromo cAMP (2mM) significantly stimulated prolactin secretion and both compounds increased growth hormone [GH] release. Cholera toxin (3  $\mu$ g/ml for 4 h) increased pituitary cAMP concentrations 4-to 5-fold, but had no effect on prolactin secretion. The inhibition of prolactin secretion by dopamine was unaffected by cholera toxin, despite the fact that dopamine had no effect on the raised pituitary cAMP concentration caused by this factor. Dopamine had no significant effect on either basal or stimulated GH secretion under any of the conditions tested. The inhibitory effects of dopamine on prolactin secretion are probably not mediated by lowering of cAMP concentration, although modulation of the concentration of this nucleotide, in some other circumstances, may alter the secretion of the hormone.

ST Major Concepts

Endocrine System (Chemical Coordination and Homeostasis); Metabolism; Nervous System (Neural Coordination); Pharmacology

ST Miscellaneous Descriptors

AUTONOMIC-DRUG THEOPHYLLINE ISO BUTYLMETHYL XANTHINE ENZYME  
INHIBITOR-DRUG DI BUTYRYL CYCLIC AMP 8 BROMO CYCLIC AMP CHOLERA TOXIN  
METABOLIC-DRUG PHOSPHO DI ESTERASE GROWTH HORMONE  
PHARMACODYNAMICS

ORGN Classifier

Vibrionaceae 06704

Super Taxa

Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria; Microorganisms

Taxa Notes

Bacteria, Eubacteria, Microorganisms

ORGN Classifier

Theaceae 26845

Super Taxa

Dicotyledones; Angiospermae; Spermatophyta; Plantae

Taxa Notes

Angiosperms, Dicots, Plants, Spermatophytes, Vascular Plants

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

RN 51-61-6 (DOPAMINE)

9002-62-4 (PROLACTIN)

60-92-4 (CYCLIC AMP)

58-55-9 (THEOPHYLLINE)

28822-58-4 (ISOBUTYLMETHYLYXANTHINE)

362-74-3 (DIBUTYRYL CYCLIC AMP)

9025-82-5 (PHOSPHODIESTERASE)

9002-72-6 (GROWTH HORMONE)

AB

The effects of dopamine on pituitary prolactin secretion and pituitary cAMP accumulation were studied by using anterior pituitary glands from adult female rats, incubated in vitro. During 2 h incubations, significant inhibition of prolactin secretion was achieved at concentrations between 1 and 10 nM-dopamine. However, 0.1-1 $\mu$ M-dopamine was required before a significant decrease in pituitary cAMP content was observed. In the presence of 1  $\mu$ M-dopamine, pituitary cAMP content decreased rapidly to reach about 75% of the control value within 20 min, and there was no further decrease for at least 2 h. Incubation with the phosphodiesterase inhibitors, theophylline (8 mM) or isobutylmethylxanthine (2mM), increased pituitary cAMP concentrations 3- and 6-fold, respectively. Dopamine (1  $\mu$ M) had no effect on the cAMP accumulation measured in the presence of theophylline, but inhibited the isobutylmethylxanthine-induced increase by 50%. The dopamine inhibition



**dopamine-induced neurotoxicity in human and rodent neuronal cultures: possible implications for Parkinson's disease**

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=> s l6 and @py<1991
'1991' NOT A VALID FIELD CODE
      0 @PY<1991
L7      0 L6 AND @PY<1991
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=> s l6 and @py=<1991
'1991' NOT A VALID FIELD CODE
      0 @PY=<1991
L8      0 L6 AND @PY=<1991
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=> s l6 and py=<1991
      14083918 PY=<1991
L9      44 L6 AND PY=<1991
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=> d his
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(FILE 'HOME' ENTERED AT 13:22:16 ON 12 SEP 2005)

FILE 'CAPLUS' ENTERED AT 13:22:39 ON 12 SEP 2005

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L1      0 S DYER/AU AND VOL AND 102 OR MOGER/AU AND VOL 97 AND 1982
L2      0 S DYER AND VOL AND 102 OR MOGER AND VOL 97 AND 1982
L3      0 S DYER.INV AND VOL AND 102 OR MOGER.INV AND VOL 97 AND 1982
      E DYER
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FILE 'CA' ENTERED AT 13:26:08 ON 12 SEP 2005

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L4      0 S (DYER/INV OR DYER/AU) AND VOL 102 AND 1985
      E DYER
L5      490 S (GROWTH OR ENHANCE OR ENHANCING) AND (CATECHOLAMINE OR EPINEP
L6      105 S (GROWTH OR ENHANCE OR ENHANCING) AND (CATECHOLAMINE OR EPINEP
L7      0 S L6 AND @PY<1991
L8      0 S L6 AND @PY=<1991
L9      44 S L6 AND PY=<1991
```

```
=> d ti 1-44
```

```
L9 ANSWER 1 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Angiotensin II increases catecholamine release from bovine
adrenal medulla but does not enhance that evoked by potassium
depolarization or by carbachol
```

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L9 ANSWER 2 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Effect of dopamine and bromocriptine on secretion of
growth hormone by pituitary growth hormone secreting
tumor in cell culture
```

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L9 ANSWER 3 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Serotonin and nialamide differentially regulate survival and
growth of cultured serotonin and catecholamine neurons
```

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L9 ANSWER 4 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Pertussis toxin stimulation of catecholamine release from
adrenal medullary chromaffin cells: mechanism may be by direct activation
of L-type and G-type calcium channels
```

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L9 ANSWER 5 OF 44 CA COPYRIGHT 2005 ACS on STN
TI Growth of cultured human cerebral meningiomas is inhibited by
dopaminergic agents. Presence of high affinity dopamine-D1
receptors
```

```
L9 ANSWER 6 OF 44 CA COPYRIGHT 2005 ACS on STN
```

L6 ANSWER 1 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Method of in vitro differentiation of neural stem cells, motor neurons, and dopamine neurons from primate embryonic stem cells

L6 ANSWER 2 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Transactivation of epidermal growth factor receptor mediates catecholamine-induced growth of vascular smooth muscle

L6 ANSWER 3 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Does acetylcholinesterase inhibition affect catecholamine secretion by adrenomedullary cells?

L6 ANSWER 4 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Role of calcium in neurotensin-evoked enhancement in firing in mesencephalic dopamine neurons

L6 ANSWER 5 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Dopamine Agonist 3-PPP Fails to Protect Against MPTP-Induced Toxicity

L6 ANSWER 6 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Myocyte contractile activity modulates norepinephrine cytotoxicity and survival effects of neuregulin-1 $\beta$

L6 ANSWER 7 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Catecholamine-induced vascular wall growth is dependent on generation of reactive oxygen species

L6 ANSWER 8 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Effects of dopamine agonists bromocriptine, pergolide, cabergoline, and SKF-38393 on GDNF, NGF, and BDNF synthesis in cultured mouse astrocytes

L6 ANSWER 9 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Role of epinephrine stimulation of CNS  $\alpha$ 1-adrenoceptors in motor activity in mice

L6 ANSWER 10 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Interactions of cyclic adenosine monophosphate, brain-derived neurotrophic factor, and glial cell line-derived neurotrophic factor treatment on the survival and growth of postnatal mesencephalic dopamine neurons in vitro

L6 ANSWER 11 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI D1 dopamine receptor regulation of cell cycle in FGF- and EGF-supported primary cultures of embryonic cerebral cortical precursor cells

L6 ANSWER 12 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI A site-specific mutation of tyrosine hydroxylase reduces feedback inhibition by dopamine in genetically modified cells grafted in parkinsonian rats

L6 ANSWER 13 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Dopamine neurons heterozygous for the Nurrl-null allele have reduced survival in vitro

L6 ANSWER 14 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Will embryonic stem cells be a useful source of dopamine neurons for transplant into patients with Parkinson's disease?

L6 ANSWER 15 OF 105 CA COPYRIGHT 2005 ACS on STN  
 TI Protective effect of insulin-like-growth-factor-1 against

- TI Chronic levodopa impairs morphological development of grafted embryonic dopamine neurons
- L9 ANSWER 7 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Differential coupling with pertussis toxin-sensitive G proteins of dopamine and somatostatin receptors involved in regulation of adenohipophyseal secretion
- L9 ANSWER 8 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Normal and adenomatous human pituitaries secrete thyrotropin-releasing hormone in vitro: modulation by dopamine, haloperidol, and somatostatin
- L9 ANSWER 9 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Long-term culture of rat mammothroph and somatotroph subpopulations separated on continuous Percoll density gradients: effects of dopamine, TRH, GHRH and somatostatin
- L9 ANSWER 10 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI A muscle-derived factor(s) induces expression of a catecholamine phenotype in neurons of cultured rat cerebral cortex
- L9 ANSWER 11 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI The effect of epinephrine and benzalkonium chloride on cultured corneal endothelial and trabecular meshwork cells
- L9 ANSWER 12 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Dihydropyridine-sensitive calcium channel activity related to prolactin, growth hormone, and luteinizing hormone release from anterior pituitary cells in culture: interactions with somatostatin, dopamine, and estrogens
- L9 ANSWER 13 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Enhancement of the response of hen granulosa cells to LH with norepinephrine in vitro
- L9 ANSWER 14 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Evidence that prostaglandins activate calcium channels to enhance basal and stimulation-evoked catecholamine release from bovine adrenal chromaffin cells in culture
- L9 ANSWER 15 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Survival and function of dissociated rat dopamine neurons grafted at different developmental stages or after being cultured in vitro
- L9 ANSWER 16 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Modification by pertussis toxin of the responses of bovine anterior pituitary cells to acetylcholine and dopamine: effects on hormone secretion and rubidium-86 efflux
- L9 ANSWER 17 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Effects of retinoic acid on alkaline phosphatase messenger ribonucleic acid, catecholamine receptors, and G proteins in ROS 17/2.8 cells
- L9 ANSWER 18 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Survival, morphology, and catecholamine storage of chromaffin cells in serum-free culture: evidence for a survival and differentiation promoting activity in medium conditioned by purified chromaffin cells
- L9 ANSWER 19 OF 44 CA COPYRIGHT 2005 ACS on STN
- TI Differential effects of nerve growth factor and ciliary neuronotrophic factor on catecholamine storage and

**catecholamine synthesizing enzymes of cultured rat chromaffin cells**

- L9 ANSWER 20 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Evidence that stimulation of **growth hormone release by epinephrine** and vasoactive intestinal peptide is based on **cell-to-cell communication** in the pituitary
- L9 ANSWER 21 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI  $\alpha$ 2-Adrenoceptors do not regulate **catecholamine secretion** by bovine adrenal medullary cells: a study with clonidine
- L9 ANSWER 22 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Role of **cell-to-cell communication** in the **growth hormone response to epinephrine, growth hormone releasing factor** and vasoactive intestinal peptide
- L9 ANSWER 23 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI **Catecholamine** modulation of embryonic palate mesenchymal cell DNA synthesis
- L9 ANSWER 24 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Relationship between **dopamine** content and its secretion in PC12 cells as a function of **cell growth**
- L9 ANSWER 25 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Effects of **growth hormone-releasing factor(1-44)** on **growth hormone release** from human somatotropinomas in vitro: interaction with somatostatin, **dopamine**, vasoactive intestinal peptide and cycloheximide
- L9 ANSWER 26 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Effect of extracellular matrix on PC 12 **cell shape** and **dopamine** processing
- L9 ANSWER 27 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Inhibition of baboon marrow CFU-GEMM, CFU-GM, BFU-E and CFU-E by adrenochrome, an **epinephrine** metabolite
- L9 ANSWER 28 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Hypothyroid pituitary cells in **culture**: an analysis of thyrotropin and prolactin responses to **dopamine (DA)** and DA receptor binding
- L9 ANSWER 29 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Effects of ascorbic acid, dexamethasone, and insulin on the **catecholamine** and opioid peptide stores of cultured adrenal medullary chromaffin cells
- L9 ANSWER 30 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Presence of nerve **growth factor** receptors and **catecholamine** uptake in subpopulations of chick sympathetic neurons: correlation with survival factor requirements in **culture**
- L9 ANSWER 31 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Angiotensin II stimulates changes in the **norepinephrine** content of primary cultures of rat brain
- L9 ANSWER 32 OF 44 CA COPYRIGHT 2005 ACS on STN  
TI Suppression of **catecholamine** and melanin synthesis and promotion of cholinergic differentiation of quail neural crest cells by heart cell conditioned medium
- L9 ANSWER 33 OF 44 CA COPYRIGHT 2005 ACS on STN

L13 ANSWER 1 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 1  
 TI An in vitro bacterial model of cytotoxicity to living cells  
 caused by dopamine and 6-hydroxydopamine oxidation at  
 physiological pH  
 AN 1991:143080 TOXCENTER  
 CP Copyright 2005 ACS  
 DN CA11503023921H  
 TI An in vitro bacterial model of cytotoxicity to living cells  
 caused by dopamine and 6-hydroxydopamine oxidation at  
 physiological pH  
 AU Giunta, Sergio; Galeazzi, Luciano; Groppa, Giuseppe  
 CS Ist. Carattere Sci., INRCA, Rome, Italy.  
 SO Free Radical Biology & Medicine, (1991) Vol. 10, No. 5, pp.  
 297-303.  
 CODEN: FRBMEH. ISSN: 0891-5849.  
 CY ITALY  
 DT Journal  
 FS CAPLUS  
 OS CAPLUS 1991:423921  
 LA English  
 ED Entered STN: 20011116  
 Last Updated on STN: 20021008  
 TI An in vitro bacterial model of cytotoxicity to living cells  
 caused by dopamine and 6-hydroxydopamine oxidation at  
 physiological pH  
 SO Free Radical Biology & Medicine, (1991) Vol. 10, No. 5, pp.  
 297-303.  
 CODEN: FRBMEH. ISSN: 0891-5849.  
 AB The cytotoxicity of dopamine (DA) and 6-hydroxydopamine (6-OHDA) on living  
 cells, in vitro, has been previously investigated in  
 neuroblastoma cells. This study was designed to explore the possibility  
 of using bacteria as targets for studying DA and 6-HODA  
 cytotoxicity. Both DA and 6-HODA oxidize when added to bacteriol. media.  
 The rate of autoxidn. of 6-HODA was greater than DA within the first  
 hours. The oxidation-dependent cytotoxicity caused bacterial growth  
 -inhibition and killing at concentration of 10<sup>-4</sup>M. All the bacterial strains  
 tested were slightly more susceptible to DA than to 6-HODA. Antioxidants  
 (sodium metabisulfite, cysteine) prevented the oxidation and abolished the  
 growth-inhibitory activity. The addition of exogenous catalase  
 protected the cells against the effect of the oxidation of both the  
 catecholamines up to the concentration of 5 mM, while the addition of exogenous  
 superoxide dismutase protected the cells only at the minimal inhibitory  
 concns. Taking into account that some of the results obtained are similar  
 to those previously reported using neuroblastoma cells as targets, the use  
 of bacteria for studying oxygen toxicity from these  
 catecholamines seems to be a potentially useful model system.  
 ST Miscellaneous Descriptors  
 dopamine oxidn cytotoxicity bacteria oxygen radical;  
 hydroxydopamine oxidn cytotoxicity bacteria oxygen radical  
 AB The cytotoxicity of dopamine (DA) and 6-hydroxydopamine (6-OHDA) on living  
 cells, in vitro, has been previously investigated in  
 neuroblastoma cells. This study was designed to explore the possibility  
 of using bacteria as targets for studying DA and 6-HODA  
 cytotoxicity. Both DA and 6-HODA oxidize when added to bacteriol. media.  
 The rate of autoxidn. of 6-HODA was greater than DA within the first  
 hours. The oxidation-dependent cytotoxicity caused bacterial growth  
 -inhibition and killing at concentration of 10<sup>-4</sup>M. All the bacterial strains  
 tested were slightly more susceptible to DA than to 6-HODA. Antioxidants  
 (sodium metabisulfite, cysteine) prevented the oxidation and abolished the  
 growth-inhibitory activity. The addition of exogenous catalase  
 protected the cells against the effect of the oxidation of both the  
 catecholamines up to the concentration of 5 mM, while the addition of exogenous  
 superoxide dismutase protected the cells only at the minimal inhibitory

concns. Taking into account that some of the results obtained are similar to those previously reported using neuroblastoma cells as targets, the use of bacteria for studying oxygen toxicity from these catecholamines seems to be a potentially useful model system.

- L13 ANSWER 2 OF 10 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 2
- TI Inhibition of human immunodeficiency virus type 1 replication and cytopathicity by synthetic soluble catecholamine melanins in vitro
- AN 1990:33630 TOXCENTER
- DN PubMed ID: 2327999
- TI Inhibition of human immunodeficiency virus type 1 replication and cytopathicity by synthetic soluble catecholamine melanins in vitro
- AU Montefiori D C; Modliszewski A; Shaff D I; Zhou J
- CS Department of Pathology, Vanderbilt University Medical School, Nashville, Tennessee 37232
- SO Biochemical and biophysical research communications, (1990 Apr 16 ) 168 (1) 200-5.  
Journal Code: 0372516. ISSN: 0006-291X.
- CY United States
- DT Journal; Article; (JOURNAL ARTICLE)
- FS MEDLINE
- OS MEDLINE 90226360
- LA English
- ED Entered STN: 20011116  
Last Updated on STN: 20011116
- TI Inhibition of human immunodeficiency virus type 1 replication and cytopathicity by synthetic soluble catecholamine melanins in vitro
- SO Biochemical and biophysical research communications, (1990 Apr 16 ) 168 (1) 200-5.  
Journal Code: 0372516. ISSN: 0006-291X.
- AB Synthetic soluble melanins were synthesized by spontaneous oxidation of L-dopamine, norepinephrine or 5-hydroxytryptamine (serotonin) in weak alkaline solution. These three melanins inhibited infection of human CD4+ lymphoblastoid cells (MT-2) by cell-free human immunodeficiency virus type 1 (HIV-1), without cell toxicity, at concentrations of 0.15-10 micrograms/ml. Also, syncytium formation and resulting cytopathic effects when uninfected cells were mixed with chronic HIV-1-infected cells were blocked by these melanins. Antisyncytial activity was greater when infected cells were preincubated with melanin than when uninfected cells were preincubated with melanin, thus suggesting that interaction of melanin with viral proteins is an important aspect of the antiviral mechanism. These results make synthetic soluble melanins interesting candidates for further study as possible anti-HIV-1 therapeutics.
- CT Check Tags: In Vitro  
\*Catecholamines: PD, pharmacology  
Cell Fusion: DE, drug effects  
Cytopathogenic Effect, Viral: DE, drug effects  
HIV-1: DE, drug effects  
\*HIV-1: GD, growth & development  
Humans  
\*Melanins: PD, pharmacology  
\*Virus Replication: DE, drug effects
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 AN 101:66401 CA  
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 AU Foord, Steven M.; Peters, John R.; Dieguez, Carlos; Jasani, Barhat; Hall, Reginald; Scanlon, Maurice F.  
 CS Dep. Med., Welsh Natl. Sch. Med., Heath Park/Cardiff, CF4 4XN, UK  
 SO Endocrinology (1984), 115(1), 407-15  
 CODEN: ENDOAO; ISSN: 0013-7227  
 DT Journal  
 LA English  
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 AB After 3 days in culture, the anterior pituitary (AP) cells from hypothyroid rats showed greater TSH [9002-71-5] and prolactin (PRL) [9002-62-4] secretory activity and less growth hormone (GH) [9002-72-6] secretory activity than did parallel euthyroid cultures. Bromocriptine [25614-03-3], apomorphine [58-00-4], and dopamine (DA) [51-61-6] inhibited euthyroid TSH secretion by .apprx.30%, whereas each drug inhibited hypothyroid TSH secretion by .apprx.60%. In contrast, the 3 agonists were less effective in inhibiting PRL secretion from hypothyroid cells. The rank order of potency [bromocriptine > (+)butaclamol [56245-67-1] > apomorphine > DA > (-)butaclamol [51152-91-1] shown against secretion was the same for TSH and PRL in both euthyroid and hypothyroid cell cultures and is typical of a DA receptor-mediated process. The binding of [3H]dihydroergocryptine(DHE) to DA receptors on euthyroid and hypothyroid cells was examined. One micromolar concentration of (+)butaclamol was used to define nonspecific binding.

#### Specific

binding was saturable and stereospecific in each case. The rank order of potency of dopaminergic agonists and antagonists in competing for [3H]DHE binding was the same as that demonstrated against the secretion of TSH and PRL. Each compound displayed more [3H]DHE from hypothyroid cells than from euthyroid cells. Construction of adsorption isotherms for [3H]DHE binding DA receptors on euthyroid and hypothyroid cells and subsequent Scatchard anal. revealed a 3-4-fold increase in receptor number without a change in affinity. Immunohistochem. on AP lobes before and after dispersion revealed an increase in thyrotrophs and thyroidectomy cells in hypothyroid rats relative to those in control animals. In euthyroid animals thyrotrophs were 10.1% of the total AP cell population, in hypothyroid animals thyrotrophs plus the thyroidectomy cells were 36.3% of the total AP cells. Therefore, the increased number of DA receptors per lobe could be accounted for by increased nos. of thyrotrophs. The mechanism of the altered sensitivity to DA induced by hypothyroidism in lactotrophs and thyrotrophs remains to be clarified.

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